```
=> kadyk /au
L1             0 FILE AGRICOLA
L2             0 FILE BIOTECHNO
L3             0 FILE CONFSCI
L4             0 FILE HEALSAFE
L5             0 FILE LIFESCI
L6             0 FILE PASCAL

TOTAL FOR ALL FILES
L7             0 KADYK

=> Lickteig k/au
L8             0 FILE AGRICOLA
L9             1 FILE BIOTECHNO
L10            0 FILE CONFSCI
L11            0 FILE HEALSAFE
L12            2 FILE LIFESCI
L13            3 FILE PASCAL

TOTAL FOR ALL FILES
L14            6 LICKTEIG K/AU

=> l14 and MAPK
L15            0 FILE AGRICOLA
L16            0 FILE BIOTECHNO
L17            0 FILE CONFSCI
L18            0 FILE HEALSAFE
L19            0 FILE LIFESCI
L20            0 FILE PASCAL

TOTAL FOR ALL FILES
L21            0 L14 AND MAPK

=> Costa m/au
L22           87 FILE AGRICOLA
L23          141 FILE BIOTECHNO
L24          149 FILE CONFSCI
L25           21 FILE HEALSAFE
L26          297 FILE LIFESCI
L27          694 FILE PASCAL

TOTAL FOR ALL FILES
L28         1389 COSTA M/AU

=> l28 and MAPK
L29            0 FILE AGRICOLA
L30            0 FILE BIOTECHNO
L31            0 FILE CONFSCI
L32            0 FILE HEALSAFE
L33            2 FILE LIFESCI
L34            0 FILE PASCAL

TOTAL FOR ALL FILES
L35            2 L28 AND MAPK

=> d l35 ibib abs total

L35  ANSWER 1 OF 2  LIFESCI    COPYRIGHT 2010 CSA on STN
ACCESSION NUMBER:   2010:9752  LIFESCI
```

TITLE: A genome-wide screen in Saccharomyces cerevisiae reveals pathways affected by arsenic toxicity
AUTHOR: Zhou, X.; Arita, A.; Ellen, T.P.; Liu, X.; Bai, J.; Rooney, J.P.; Kurtz, A.D.; Klein, C.B.; Dai, W.; Begley, T.J.; Costa, M.
CORPORATE SOURCE: New York University School of Medicine, 57 Old Forge Road, Tuxedo, NY 10987, USA; E-mail: max.costayumc.org
SOURCE: Genomics, (20091100) vol. 94, no. 5, pp. 294-307. ISSN: 0888-7543.
DOCUMENT TYPE: Journal
FILE SEGMENT: G; K
LANGUAGE: English
SUMMARY LANGUAGE: English

AB We have used Saccharomyces cerevisiae to identify toxicologically important proteins and pathways involved in arsenic-induced toxicity and carcinogenicity in humans. We performed a systemic screen of the complete set of 4733 haploid S. cerevisiae single-gene-deletion mutants to identify those that have decreased or increased growth, relative to wild type, after exposure to sodium arsenite (NaAsO sub(2)). IC sub(5) sub(0) values for all mutants were determined to further validate our results. Ultimately we identified 248 mutants sensitive to arsenite and 5 mutants resistant to arsenite exposure. We analyzed the proteins corresponding to arsenite-sensitive mutants and determined that they belonged to functional categories that include protein binding, phosphate metabolism, vacuolar/lysosomal transport, protein targeting, sorting, and translocation, cell growth /morphogenesis, cell polarity and filament formation. Furthermore, these data were mapped onto a protein interactome to identify arsenite-toxicity-modulating networks. These networks are associated with the cytoskeleton, ubiquitination, histone acetylation and the MAPK signaling pathway. Our studies have potential implications for understanding toxicity and carcinogenesis in arsenic-induced human conditions, such as cancer and aging.

L35 ANSWER 2 OF 2 LIFESCI COPYRIGHT 2010 CSA on STN
ACCESSION NUMBER: 2005:119254 LIFESCI
TITLE: Differential effects of polycyclic aromatic hydrocarbons on transactivation of AP-1 and NF- Kappa B in mouse epidermal cl41 cells
AUTHOR: Li, Jingxia; Chen, Haobin; Ke, Qingdong; Feng, Zhaohui; Tang, Moon-Shong; Liu, B.; Amin, S.; Costa, M.; Huang, Chuanshu
CORPORATE SOURCE: Nelson Institute of Environmental Medicine, New York University School of Medicine, 57 Old Forge Road, Tuxedo, NY 10987, USA
SOURCE: Molecular Carcinogenesis [Mol. Carcinog.], (20040600) vol. 40, no. 2, pp. 104-115. ISSN: 0899-1987.
DOCUMENT TYPE: Journal
FILE SEGMENT: X
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Polycyclic aromatic hydrocarbons (PAHs) and their derivatives, such as benzo[a]pyrene (B[a]P), (plus or minus)-anti-benzo[a]pyrene-7,8-diol-9,10-epoxide (B[a]PDE), and 5-methylchrysene-1,2-diol-3,4-epoxide (5-MCDE), are complete carcinogens. However, the tumor promotion effects of PAHs remain unclear. We therefore investigated the possible activation of activator protein-1 (AP-1) and nuclear factor- Kappa B (NF Kappa B) in mouse epidermal C141 cells after different PAHs treatments, including B[a]P, B[a]PDE, chrysene-1,2-diol-3,4- epoxid (CDE), and 5-MCDE. The results showed that B[a]PDE and 5-MCDE were able to activate AP-1 and NF- Kappa B, whereas

B[a]P showed only marginal effect on AP-1 activation, and B[a]P and CDE had no effect on NF- Kappa B activation. Treatment with either B[a]PDE or 5-MCDE also resulted in mitogen-activated protein kinases (MAPKs) activation as well as inhibitory subunit kappa-B (I Kappa B alpha) phosphorylation and degradation, whereas B[a]P and CDE had no effect. Pretreatment with PD98059, a specific inhibitor for extracellular signal-regulated protein kinases (ERKs) upstream kinase MEK1/2, or SB202190, a p38 kinase inhibitor, resulted in a dramatic inhibition of B[a]PDE-induced AP-1 transactivation. In addition, B[a]PDE- induced AP-1 activation was also inhibited by overexpressing a dominant negative mutant of JNK1 in the cells. All these suggest ERKs, c-jun N- terminal kinases (JNKs), and p38 kinase signal transduction pathways are required for AP-1 induction by B[a]PDE. Taken together, B[a]PDE and 5-MCDE are the active compounds of PAHs to initiate signaling pathways. Considering the important roles of AP-1 and NF- Kappa B in tumor promotion, we speculated the activation of AP-1 and NF- Kappa B by B[a]PDE and 5-MCDE may involve in their or their parent compounds' tumor promotion effects. This study may help in better understanding the tumor promotion effects of PAHs.

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=> MAPK and (genetic screen)
L36           1 FILE AGRICOLA
L37          15 FILE BIOTECHNO
L38           0 FILE CONFSCI
L39           0 FILE HEALSAFE
L40          30 FILE LIFESCI
L41           3 FILE PASCAL

TOTAL FOR ALL FILES
L42          49 MAPK AND (GENETIC SCREEN)

=> 142 and (rac or axin or beta-catenin)
L43           0 FILE AGRICOLA
L44           0 FILE BIOTECHNO
L45           0 FILE CONFSCI
L46           0 FILE HEALSAFE
L47           0 FILE LIFESCI
L48           0 FILE PASCAL

TOTAL FOR ALL FILES
L49           0 L42 AND (RAC OR AXIN OR BETA-CATENIN)

=> (rac or axin or beta-catenin) and MAPK
L50          13 FILE AGRICOLA
L51          81 FILE BIOTECHNO
L52           1 FILE CONFSCI
L53           0 FILE HEALSAFE
L54         171 FILE LIFESCI
L55          98 FILE PASCAL

TOTAL FOR ALL FILES
L56         364 (RAC OR AXIN OR BETA-CATENIN) AND MAPK

=> 156 and screen
L57           1 FILE AGRICOLA
L58           1 FILE BIOTECHNO
L59           0 FILE CONFSCI
L60           0 FILE HEALSAFE
L61           3 FILE LIFESCI
L62           0 FILE PASCAL
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TOTAL FOR ALL FILES
L63 5 L56 AND SCREEN

=> dup rem
ENTER L# LIST OR (END):163
PROCESSING COMPLETED FOR L63
L64 4 DUP REM L63 (1 DUPLICATE REMOVED)

=> d l64 ibib abs total

L64 ANSWER 1 OF 4 LIFESCI COPYRIGHT 2010 CSA on STN
ACCESSION NUMBER: 2009:458540 LIFESCI
TITLE: An integrated genome screen identifies the Wnt signaling pathway as a major target of WT1
AUTHOR: Kim, Marianne K.-H.; McGarry, Thomas J.; " Broin, Pilib; Flatow, Jared M.; Golden, Aaron A.-J.; Licht, Jonathan D.
CORPORATE SOURCE: E-mail: j-lichtorthwestern.edu
SOURCE: Proceedings of the National Academy of Sciences, USA [Proc. Natl. Acad. Sci. USA], (20090700) vol. 106, no. 27, pp. 11154-11159.
ISSN: 0027-8424.
DOCUMENT TYPE: Journal
FILE SEGMENT: G; B
LANGUAGE: English
SUMMARY LANGUAGE: English
AB WT1, a critical regulator of kidney development, is a tumor suppressor for nephroblastoma but in some contexts functions as an oncogene. A limited number of direct transcriptional targets of WT1 have been identified to explain its complex roles in tumorigenesis and organogenesis. In this study we performed genome-wide screening for direct WT1 targets, using a combination of ChIP-ChIP and expression arrays. Promoter regions bound by WT1 were highly G-rich and resembled the sites for a number of other widely expressed transcription factors such as SP1, MAZ, and ZNF219. Genes directly regulated by WT1 were implicated in MAPK signaling, axon guidance, and Wnt pathways. Among directly bound and regulated genes by WT1, nine were identified in the Wnt signaling pathway, suggesting that WT1 modulates a subset of Wnt components and responsive genes by direct binding. To prove the biological importance of the interplay between WT1 and Wnt signaling, we showed that WT1 blocked the ability of Wnt8 to induce a secondary body axis during Xenopus embryonic development. WT1 inhibited TCF-mediated transcription activated by Wnt ligand, wild type and mutant, stabilized beta -catenin by preventing TCF4 loading onto a promoter. This was neither due to direct binding of WT1 to the TCF binding site nor to interaction between WT1 and TCF4, but by competition of WT1 and TCF4 for CBP. WT1 interference with Wnt signaling represents an important mode of its action relevant to the suppression of tumor growth and guidance of development.

L64 ANSWER 2 OF 4 AGRICOLA Compiled and distributed by the National Agricultural Library of the Department of Agriculture of the United States of America. It contains copyrighted materials. All rights reserved. (2010) on STN
ACCESSION NUMBER: 2007:15596 AGRICOLA
DOCUMENT NUMBER: IND43870677
TITLE: SCLIP, a Microtubule-destabilizing Factor, Interacts with RasGRF1 and Inhibits Its Ability to Promote Rac Activation and Neurite Outgrowth.
AUTHOR(S): Baldassa, Simona; Gnesutta, Nerina; Fascio, Umberto; Sturani, Emmapaola; Zippel, Renata
SOURCE: Journal of biological chemistry, 2007 Jan. 26 Vol. 282, no. 4 p. 2333-2345

Publisher: American Society for Biochemistry and Molecular Biology
ISSN: 0021-9258
NOTE: Includes references
DOCUMENT TYPE: Article; (ELECTRONIC RESOURCE)
FILE SEGMENT: Other US
LANGUAGE: English

AB RasGRF1 is a neuron-specific guanine nucleotide exchange factor for the small GTPases Ras and Rac. It is implicated in the regulation of memory formation and in the development of tolerance to drug abuse, although the mechanisms have been elucidated only in part. Here we report the isolation, by the yeast two-hybrid screen, of the microtubule-destabilizing factor SCLIP (SCG10-like protein) as a novel RasGRF1-interacting protein. This interaction requires the region spanning the Dblhomology domain of RasGRF1, endowed with catalytic activity on Rac. In search for a possible function we found by biochemical means that SCLIP influences the signaling properties of RasGRF1, greatly reducing its ability to activate the Rac/p38 MAPK pathway, while the Ras/Erk one remains unaffected. Moreover, a potential role is suggested by transfection studies in neuronal PC12 cells in which RasGRF1 induces neurite outgrowth, and coexpression of SCLIP counteracts this effect, causing a dramatic decrease in the percentage of cells bearing neurites, which also appear significantly shortened. This study unveils a physical and functional interaction between RasGRF1 and SCLIP. We suggest that this novel interplay may have possible implications in mechanisms that regulate neuronal morphology and structural plasticity.

L64 ANSWER 3 OF 4 LIFESCI COPYRIGHT 2010 CSA on STN
ACCESSION NUMBER: 2004:92320 LIFESCI
TITLE: A role for MKP3 in axial patterning of the zebrafish embryo
AUTHOR: Tsang, M.; Maegawa, S.; Kiang, A.; Habas, R.; Weinberg, E.; Dawid, I.B.
CORPORATE SOURCE: Laboratory of Molecular Genetics, National Institute of Child Health and Human Development, National Institutes of Health, Bethesda, MD 20892, USA; E-mail: idawid@nih.gov
SOURCE: Development, (20040615) vol. 131, no. 12, pp. 2769-2779. ISSN: 0950-1991.
DOCUMENT TYPE: Journal
FILE SEGMENT: G
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Fibroblast growth factors (FGFs) are secreted molecules that can activate the RAS/mitogen-activated protein kinase (MAPK) pathway to serve crucial functions during embryogenesis. Through an in situ hybridization screen for genes with restricted expression patterns during early zebrafish development, we identified a group of genes that exhibit similar expression patterns to FGF genes. We report the characterization of zebrafish MAP kinase phosphatase 3 (MKP3; DUSP6--Zebrafish Information Network), a member of the FGF synexpression group, showing that it has a crucial role in the specification of axial polarity in the early zebrafish embryo. MKP3 dephosphorylates the activated form of MAPK, inhibiting the RAS/MAPK arm of the FGF signaling pathway. Gain- and loss-of-function studies reveal that MKP3 is required to limit the extent of FGF/RAS/MAPK signaling in the early embryo, and that disturbing this inhibitory pathway disrupts dorsoventral patterning at the onset of gastrulation. The earliest mkp3 expression is restricted to the future dorsal region of the embryo where it is initiated by a maternal beta -catenin signal, but soon after its initiation, mkp3 expression comes under the control of FGF signaling. Thus, mkp3 encodes a feedback attenuator of the FGF pathway, the expression of which is initiated at an early stage so as to ensure correct FGF signaling

levels at the time of axial patterning.

L64 ANSWER 4 OF 4 BIOTECHNO COPYRIGHT 2010 Elsevier Science B.V. on STN DUPLICATE

ACCESSION NUMBER: 1999:29358561 BIOTECHNO
TITLE: Cellular functions of TC10, a Rho family GTPase: Regulation of morphology, signal transduction and cell growth
AUTHOR: Murphy G.A.; Solski P.A.; Jillian S.A.; De la Ossa P.P.; D'Eustachio P.; Der C.J.; Rush M.G.
CORPORATE SOURCE: M.G. Rush, Department of Biochemistry, NYU Medical Center, 550 First Avenue, New York, NY 10016, United States.
SOURCE: Oncogene, (01 JUL 1999), 18/26 (3831-3845), 37 reference(s)
CODEN: ONCNES ISSN: 0950-9232
DOCUMENT TYPE: Journal; Article
COUNTRY: United Kingdom
LANGUAGE: English
SUMMARY LANGUAGE: English

AN 1999:29358561 BIOTECHNO
AB The small Ras-related GTPase, TC10, has been classified on the basis of sequence homology to be a member of the Rho family. This family, which includes the Rho, Rac and CDC42 subfamilies, has been shown to regulate a variety of apparently diverse cellular processes such as actin cytoskeletal organization, mitogen-activated protein kinase (MAPK) cascades, cell cycle progression and transformation. In order to begin a study of TC10 biological function, we expressed wild type and various mutant forms of this protein in mammalian cells and investigated both the intracellular localization of the expressed proteins and their abilities to stimulate known Rho family-associated processes. Wild type TC10 was located predominantly in the cell membrane (apparently in the same regions as actin filaments), GTPase defective (75L) and GTP-binding defective (31N) mutants were located predominantly in cytoplasmic perinuclear regions, and a deletion mutant lacking the carboxyl terminal residues required for posttranslational prenylation was located predominantly in the nucleus. The GTPase defective (constitutively active) TC10 mutant: (1) stimulated the formation of long filopodia; (2) activated c-Jun amino terminal kinase (JNK); (3) activated serum response factor (SRF)-dependent transcription; (4) activated NF-κB-dependent transcription; and (5) synergized with an activated Raf-kinase (Raf-CAAX) to transform NIH3T3 cells. In addition, wild type TC10 function is required for full H-Ras transforming potential. We demonstrate that an intact effector domain and carboxyl terminal prenylation signal are required for proper TC10 function and that TC10 signals to at least two separable downstream target pathways. In addition, TC10 interacted with the actin-binding and filament-forming protein, profilin, in both a two-hybrid cDNA library screen, and an in vitro binding assay. Taken together, these data support a classification of TC10 as a member of the Rho family, and in particular, suggest that TC10 functions to regulate cellular signaling to the actin cytoskeleton andprocesses associated with cell growth.